ACIDOSIS DURING EXERTION STIMULATES THE RELEASE OF BETA-ENDORHIN.

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The release of beta-endorphin as been correlated with the degree of hypoxia in perinatally stressed infants at birth. One of the most common markers of severe hypoxia is a drop in pH due to metabolic acidosis. Similarly beta-endorphin release during exercise has been related to intensity of exertion and metabolic acidosis. Healthy adult males were stressed in exertion tasks incrementally to exhaustion, under conditions that created metabolic acidosis and conditions where acidosis was blocked in order to determine if acidosis directly stimulates the release of beta-endorphin. Each subject performed an exertion task using the Bruce protocol. Serial arterial blood samples were taken during the test and analyzed for beta-endorphin, base excess, lactate, pH, PCO_2, and PO_2. Resiratory measures RR, VE, $VC0_2$, and $V0_2$ were taken four times a minute during all tests. From these values the level of exertion at which the subject reached 85% of $V0_2$ maximum and the predicted base excess at that exertion level were calculated. Subsequently, each subject exercised at a steady state maintaining an exertion level eliciting 85% of VO_2 maximum for twenty minutes. Subjects ingested .3g/kg of sodium bicarbonate prior to exertion and were infused with an amount of an isotonic sodium bicarbonate solution equivalent to their predicted base excesses. Serial arterial samples were taken every two minutes. In a blinded control run a placebo solution was ingested and normal saline was infused. The relationship betweens acidosis, lactate, respiratory drive, and beta-endorphin samples were examined. Regression of respiratory values and blood gases versus beta-endorphin during incremental exertion indicated that change in pH best predicted beta-endorphin response. Furthermore, blood buffering with bicarbonate attenuated the endorphin response.